JACC Vol. 24, No. 1
July 1994:46–54

# Directional Coronary Atherectomy in Unstable Angina

ALAA E. ABDELMEGUID, MD, PHD,* STEPHEN G. ELLIS, MD, FACC, SHELLY K. SAPP, MS, CONRAD SIMPFENDORFER, MD, FACC, IRVING FRANCO, MD, FACC, PATRICK L. WHITLOW, MD, FACC

*Cleveland, Ohio*

***Objectives.*** **To determine whether excision of complex, ulcerated plaque improves the risk of patients with unstable angina to the level of those with stable angina, the results of directional coronary atherectomy were compared in patients with these two syndromes.**

***Background.*** **The procedural results of angioplasty in the setting of unstable angina are not as favorable as those observed for chronic stable angina, presumably because thrombus-associated plaque augments the risk of abrupt closure.**

***Methods.*** **Two hundred eighty-seven consecutive patients who had undergone directional atherectomy for a single new stenosis were studied. Seventy-seven patients had stable angina (Group I); 110 patients had progressively worsening angina in the absence of rest or postinfarction angina (Group II); and 100 patients had rest or postinfarction angina, or both (Group III).**

***Results.*** **Major ischemic complications (death, Q wave infarction, emergency bypass surgery) occurred more frequently in Group III (1.3% [Group I] vs. 0.9% [Group II] vs. 7% [Group III], p = 0.036). This difference was largely due to a higher incidence of emergency surgery in Group III (1.3% [Group I] vs. 0% [Group II] vs. 5% [Group III], p = 0.05). Clinical follow-up was obtained in 97% of successful procedures for a mean follow-up period of 22 months (range 9 to 52) and revealed a higher incidence of hospital admission for angina (p = 0.05) and a trend toward more bypass surgery (p = 0.09) and myocardial infarction (p = 0.16) in Group III. There was no difference in repeat percutaneous interventions among the three groups (range 19% to 24%, p = 0.75).**

***Conclusions.*** **These results show that the definition of unstable angina is important in determining the immediate outcome of directional atherectomy. In the absence of rest or postinfarction angina, the immediate results are not significantly different from those obtained in stable angina. Our results also suggest that both the immediate and short-term outcome in unstable angina are not greatly influenced by atherectomy but more so by the pathophysiology of unstable angina, which increases the complications of percutaneous interventions.**

***(J Am Coll Cardiol 1994;24:46–54)***

Unstable angina pectoris is increasingly becoming one of the most common indications for percutaneous revascularization (1,2). Depending on the type of practice, patients with unstable angina represent 20% to 70% of the population in reported coronary angioplasty series (1). However, despite the extensive use of angioplasty in this setting, the procedural results are not as favorable as those observed with coronary angioplasty for chronic stable angina. The initial success rates in unstable angina range from 70% to 92%, somewhat lower than the >90% success rate achieved in patients with stable angina (3,4). This is mainly due to the higher complication rates in patients with unstable angina who undergo coronary angioplasty. The presumptive explanation for increased complications has been the association of unstable angina with intracoronary thrombus, which carries an augmented risk of abrupt closure after angioplasty (5–11). The hazard of angioplasty in this setting is most likely related to the principle that "thrombus begets thrombus," particularly after mechanical disruption with a balloon catheter (12). By causing further injury of an already ulcerated plaque in unstable angina, coronary angioplasty causes increased platelet deposition, aggregation and thrombus formation, events that could culminate in abrupt vessel closure (12). Thrombus-induced spasm may also play a role in this situation (13).

Directional coronary atherectomy is a relatively new percutaneous coronary intervention that physically removes the unstable plaque and could therefore decrease these complications. Theoretically, the controlled excision of this pathologic tissue by directional atherectomy might create a smooth lumen without the dissections, flaps or thrombus that are common after angioplasty (14,15). It is conceivable, therefore, that the procedure could allow the safe treatment of complex lesions that might be difficult to approach by conventional angioplasty and leave a smooth lumen that is less conducive to platelet aggregation and thrombus formation with a potential reduction in acute complications.

From the Department of Cardiology, The Cleveland Clinic Foundation, Cleveland, Ohio.

Manuscript received November 19, 1993; revised manuscript received January 24, 1994, accepted January 28, 1994.

*Present address: Cardiology Division (IIIA), Brooklyn Veterans Affairs Medical Center, 800 Poly Place, Brooklyn, New York 11209.

Address for correspondence: Dr. Patrick L. Whitlow, Director, Interventional Cardiology, Department of Cardiology, F25, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44195.



0735-1097/94/$7.00

The aim of this study was to compare the results of directional coronary atherectomy in patients with unstable and stable angina in an attempt to verify whether excision of the unstable plaque decreases the risk and complications to levels comparable to those observed in stable angina.

## Methods

**Patients.** This study comprised all patients with attempted directional coronary atherectomy using the Simpson Coronary Atherocath (Devices for Vascular Interventions, Inc.) at The Cleveland Clinic Foundation who underwent the procedure for a de novo single lesion as their only intervention between August 1988 and June 1992. Patients with acute myocardial infarction within 36 h, those undergoing a salvage atherectomy for failed angioplasty and those with a procedure for chronic total occlusion were excluded from this analysis. All patients gave informed consent before the procedure.

Patients with stable angina as the indication for atherectomy comprised Group I. Because there is no universally accepted definition of unstable angina, and because its broad definition covers many different subgroups of patients, we separated the patients with unstable angina into two groups. One group (Group II) included patients with unstable angina (as defined later) in the absence of rest or postmyocardial infarction pain. The other group (Group III) included patients with rest angina or post myocardial infarction angina, or both (>36 h but <30 days postinfarction). Group III has been shown to be at particularly high risk for coronary angioplasty complications (3).

Directional coronary atherectomy was attempted in 287 consecutive patients who met the study criteria. Patient selection for directional atherectomy was considered if a relatively proximal and eccentric stenosis was located in a vessel of sufficient caliber to allow the passage of the atherectomy device. Seventy-seven patients had stable angina pectoris (Group I), and 210 patients had unstable angina. In the unstable angina group, 110 patients had no rest or postinfarction angina (Group II), and 100 patients had rest or postinfarction angina, or both (Group III).

**Technique of directional coronary atherectomy.** Five French to 7F atherectomy catheters were used. The coronary atherectomy procedure was performed as described in detail elsewhere (15,16). The number of cuts performed, maximal balloon pressure and the final device size were determined by the reference vessel size and residual stenosis after atherectomy. If a significant stenosis remained after the initial atherectomy pass, subsequent atherectomy was performed by using either a higher inflation pressure or a larger atherectomy device if appropriate. Predilation with conventional balloon angioplasty was performed when difficulty in crossing the lesion was anticipated, or when the atherectomy catheter could not be advanced across the stenosis. If significant stenosis remained after the atherectomy attempts, the lesion was subsequently dilated with a standard balloon to achieve an adequate result. Patients received routine care before and after directional coronary atherectomy, including pretreatment with aspirin and a calcium-channel blocking agent. Intravenous heparin (10,000 to 15,000 U) was administered at the beginning of the procedure, followed by additional boluses to maintain an activated clotting time >300 s. The duration of heparinization after the procedure was left to the physician's discretion. After completion of the procedure, the patients were monitored in an intensive care unit or a postprocedure telemetry ward. A 12-lead electrocardiogram (ECG) was routinely obtained after the procedure, on the following day and in the event of any chest pain suggesting ischemia. Cardiac enzyme levels were routinely measured after the procedure and on the following day. When the creatine kinase (CK) level was elevated, it was measured every 8 h until it returned to normal levels. Patients were maintained on aspirin, and a calcium channel blocker was administered for at least 48 h after the procedure.

**Clinical and procedural variables.** Clinical information at the time of the initial presentation and data obtained at the time of the procedure and at discharge were recorded prospectively on standard case report forms and entered in the Cleveland Clinic Interventional Registry Database. An experienced angiographer reviewed the diagnostic and procedural cineangiograms to code for lesion-related morphologic variables (eccentricity, length, angulation, bifurcation stenosis, calcification and thrombus-containing lesions). Angiographic measurements before and after directional coronary atherectomy were performed by using hand-held calipers in the projection showing the most severe stenosis, with the guiding catheter serving as the reference standard. Angiographic data were also entered prospectively in the registry data base.

**Follow-up.** Clinical follow-up data were obtained by trained interventional registry personnel who made telephone contact calls with the referral patients in this study and by visits of patients followed up at Cleveland Clinic. The patients were questioned as to the recurrence of symptoms, hospital admission for angina, repeat revascularization and myocardial infarction. Follow-up events were analyzed and classified by a physician. The families or physicians, or both, of deceased patients were interviewed in an effort to ascertain the cause of death.

**Definitions.** *Unstable angina.* Unstable angina was defined as new onset (or acceleration of preexisting) angina <1 month in duration, angina with a progressive or crescendo pattern, increasing frequency or duration of attacks or rest, or postinfarction angina (>36 h but <30 days).

*Angiographic definitions.* The angiographic definitions used in this analysis have been used in the evaluation of the results of coronary angioplasty and have been published elsewhere (17,18). Multivessel disease was defined as >70% diameter stenosis in at least one major coronary artery with >50% diameter stenosis in one or more other major coronary artery (19).

JACC Vol. 24, No. 1
July 1994:46–54

**Table 1.** Clinical Demographics of the Study Patients

| | Group I (n = 77) | Group II (n = 110) | Group III (n = 100) | p Value |
|---|---|---|---|---|
| Age (yr) | 60 | 61 | 59 | |
| % Male | 75 | 75 | 69 | |
| Angina duration (mo) | 10.5 | 3.4 | 2.8 | 0.003 |
| Previous myocardial infarction (%) | 35 | 27 | 54 | 0.002 |
| Previous CABG (%) | 16 | 29 | 33 | |
| Risk factors | | | | |
| Hypertension (%) | 47 | 49 | 53 | |
| Diabetes (%) | 22 | 15 | 24 | |
| Serum cholesterol >200 (%) | 64 | 55 | 61 | |
| Family history of CAD (%) | 49 | 65 | 65 | 0.05 |
| History of smoking (%) | 69 | 81 | 75 | |
| Former | 48 | 53 | 45 | |
| Current | 21 | 28 | 30 | |
| Multivessel disease (%) | 43 | 44 | 51 | |
| LVEF <40% | 19 | 13 | 22 | |

Data presented are number (%) of patients. CABG = coronary artery bypass graft surgery; LVEF = left ventricular ejection fraction.

**Table 2.** Lesion Morphology and Procedural Characteristics

| | Group I | Group II | Group III | p Value |
|---|---|---|---|---|
| Lesion morphology | | | | |
| Length (mm) | 7.5 | 6.6 | 5.5 | |
| Eccentric (%) | 80.5 | 70.0 | 79.0 | |
| Calcified (%) | 9.7 | 5.6 | 6.0 | |
| Bifurcation (%) | 5.1 | 2.7 | 8.0 | |
| Thrombus (%) | 2.6 | 10.0 | 7.0 | |
| Vessel (%) | | | | |
| LAD | 57.1 | 59.1 | 55 | |
| RCA | 26.0 | 13.6 | 19 | |
| Cx | 10.4 | 7.3 | 12 | |
| SVG | 6.5 | 17.3 | 12 | |
| LMT | 0 | 2.7 | 2 | |
| Diameter stenosis (%) | | | | |
| Before DCA | 76.3 | 80.2 | 79.8 | |
| After DCA | 19.1 | 14.5 | 16.8 | |
| Adjunctive percutaneous coronary angioplasty (%) | | | | |
| Before DCA | 19.5 | 18.1 | 33 | 0.025 |
| After DCA | 18.2 | 18.2 | 19 | |

Data presented are percent of patients. Cx = circumflex coronary artery; DCA = directional coronary atherectomy; LAD = left anterior descending coronary artery; LMT = left main trunk; RCA = right coronary artery; SVG = saphenous vein graft.

*Outcome variables.* Success was defined as a final percent diameter stenosis <50% (with or without ancillary balloon angioplasty), tissue removal and no major complications. Major ischemic complications were considered bypass surgery, Q wave myocardial infarction or death and were defined according to the National Heart, Lung, and Blood Institute definitions (20).

**Statistical analysis.** Statistical analysis was performed using a computerized statistical analysis program (SAS Institute Inc.). Data are expressed as mean values ± SD, unless otherwise indicated. The three groups were compared using the chi-square test or the Fisher exact test to examine differences in categoric variables. One-way analysis of variance was used to assess differences in continuous variables, with the Bonferonni correction for multiple comparisons used when pairwise comparisons were performed. Survival curves were performed using the Kaplan-Meier method with the log rank test for assessing differences in between-group outcome. A significance level of 0.05 was assumed.

## Results

**Patient profiles (Table 1).** The baseline patient information in each of the three groups is enumerated in Table 1. The study patients were congruent with those seen in other directional coronary atherectomy series (14,15,17,21). A comparison of baseline patient demographics in this series with those reported for coronary angioplasty in unstable angina (4,22) finds many similar patient characteristics but also a slightly higher incidence of multivessel disease and previous bypass surgery in our patients. The group with stable angina (Group I) had a longer duration of preintervention angina (10.5 months [Group I] vs. 3.4 months [Group II] vs. 2.8 months [Group III], p = 0.003), reflecting the high proportion of new angina in both unstable angina groups. Group III had a higher incidence of preintervention myocardial infarction (35% [Group I] vs. 27% [Group II] vs. 54% [Group III], p = 0.002), which was largely due to the inclusion of the subset of patients with postinfarction angina in Group III. Notably, multivessel disease and left ventricular dysfunction were equally distributed among the three groups.

**Morphologic and procedural characteristics (Table 2).** Limited procedural characteristics are described in Table 2. The morphologic lesion characteristics were similar to those of previously published series of directional atherectomy (14,15,17,21,23). Similar to the previously published series, most of the stenoses attempted were discrete, noncalcified and eccentric. The left anterior descending coronary artery was the most frequent target vessel, followed by the right coronary artery. There was a trend toward more thrombus-associated lesions in the unstable angina groups (p = 0.15). There was also a trend toward tighter stenoses in the unstable angina groups (p = 0.096). Preatherectomy balloon angioplasty was used more frequently in Group III (20% [Group I] vs. 18% [Group II] vs. 33% [Group III], p = 0.025), whereas the use of postatherectomy balloon dilation was seldom required in any group. There was no significant difference among the groups with regard to final residual stenosis (range 15% to 19%).

**Immediate procedural results (Table 3).** Procedural success and complication data for the different groups are presented in Table 3. Unsuccessful procedures or major

Table 3. Immediate Procedural Results

| | Group I | Group II | Group III | p Value |
|---|---|---|---|---|
| Clinical success | 76 (98.7) | 109 (99.1) | 93 (93) | 0.04 |
| Abrupt closure | 2 (2.6) | 1 (0.9) | 5 (5.0) | |
| Major complications | | | | |
| In-hospital death | 0 (0.0) | 1 (0.9) | 2 (2.0) | |
| Q wave MI | 0 (0.0) | 0 (0.0) | 1 (1.0) | |
| CABG | 1 (1.3) | 0 (0.0) | 5 (5.0) | 0.05 |
| Any event | 1 (1.3) | 1 (0.9) | 7 (7.0) | 0.04 |
| CK elevation | | | | |
| CK (IU/liter) | 150 ± 171 | 161 ± 240 | 433 ± 1,795 | |
| CK >180 IU/liter (%) | 16 | 22 | 25 | |
| Non-Q wave MI | 1 (1.3) | 2 (1.8) | 2 (2.0) | |

Data presented are mean values ± SD or number (%) of patients. CABG = coronary artery bypass graft surgery; CK = creatine kinase; MI = myocardial infarction.

ischemic complications, or both, occurred in nine patients (3.1%). Three patients died (1%) after atherectomy of the left anterior descending (two patients) and left circumflex (one patient) coronary arteries. One patient died after abrupt closure of the left main coronary artery secondary to guide catheter-induced dissection; the second patient died after abrupt closure of the target vessel, resulting in ventricular fibrillation; and the third patient died of sepsis after an apparently successful procedure. Six patients (2.1%) had emergency bypass surgery (after atherectomy of the left anterior descending coronary artery [two patients] and the right coronary artery [four patients]). Acute Q wave myocardial infarction occurred in one patient (0.3%) who underwent atherectomy of the right coronary artery. Clinical success (i.e., angiographic success in the absence of death, Q wave infarction or emergency bypass surgery) was significantly lower in Group III (98.7% [Group I] vs. 99.1% [Group II] vs. 93% [Group III], p = 0.036). There was no difference among the groups with regard to the incidence of in-hospital death or procedure-related myocardial infarction. There was a significantly higher incidence of emergency bypass surgery in Group III (1.3% [Group I] vs. 0% [Group II] vs. 5% [Group III], p = 0.05). Taking all of the major complications together (death, Q wave myocardial infarction, emergency coronary artery bypass graft), there was a significantly higher incidence of these complications in the group with rest pain or postinfarction angina (1.3% [Group I] vs. 0.9% [Group II] vs. 7% [Group III], p = 0.036).

**Abrupt vessel closure.** Abrupt in-laboratory closure occurred in two patients in Group I (2.6%). One patient was referred for emergency surgery, and the other was successfully treated with conventional balloon angioplasty. In Group II, abrupt in-laboratory closure occurred in one patient (0.9%). This patient went into ventricular fibrillation and expired, constituting the only death in this group. There were five closures in Group III (5%). One patient expired after guide catheter–induced left main coronary artery dissection, and the other four patients were referred for emergency bypass surgery.

**Creatine kinase elevation.** There was a slightly higher level of CK release after the procedure in Group III compared with the two other groups (peak CK 150 IU/liter [Group I] vs. 161 IU/liter [Group II] vs. 433 IU/liter [Group III], p = 0.12, with mean myocardial band (MB) isoenzyme of 4.0% [Group I] vs. 5.1% [Group II] vs. 4.6% [Group III]). Moreover, in Group III, 25% of patients had peak CK above the upper limit of normal (180 IU/liter), compared with 16% in Group I and 22% in Group II (p = NS). There was one non-Q wave myocardial infarction in Group I, two in Group II and two in Group III.

**Follow-up (Fig. 1 and 2).** Clinical follow-up was available in 269 (97%) of 278 patients who had a successful procedure. The mean follow-up period was 22 months (range 9 to 52). The results are shown in Figures 1 and 2. In terms of absolute events, there was a 1% to 3% mortality rate in the first year, with a 30% to 38% incidence of other events (myocardial infarction 1% to 6%; bypass surgery 4% to 12%; percutaneous coronary intervention 18% to 21%; and hospital admission for angina 22% to 30%). After the first year, there was a 3% to 5% annual mortality rate (myocardial infarction 0% to 3%; bypass surgery 0% to 2%; percutaneous coronary intervention 1% to 2%; and hospital admission for angina 1% to 4%), and all events were consistently higher in the unstable angina groups. Analysis of our long-term clinical follow-up shows that the majority of adverse events occurred within the first year after atherectomy, and consisted mainly of repeat revascularization procedures (coronary bypass 4.1% [Group I] vs. 8.6% [Group II] vs. 11.5% [Group III]; repeat percutaneous interventions 17.6% [Group I] vs. 19.9% [Group II] vs. 21.4% [Group III]). Hospital admission for angina occurred in the first year at a rate of 21.7% for Group I, 24.1% for Group II and 30.4% for Group III.

There was no difference in survival or incidence of repeat percutaneous revascularization among the three groups (Fig. 1A and D). There was a trend toward more bypass surgery (p = 0.09) and myocardial infarction (p = 0.16) in the patients with unstable angina (Fig. 1B and C). There was a

JACC Vol. 24, No. 1
July 1994:46–54

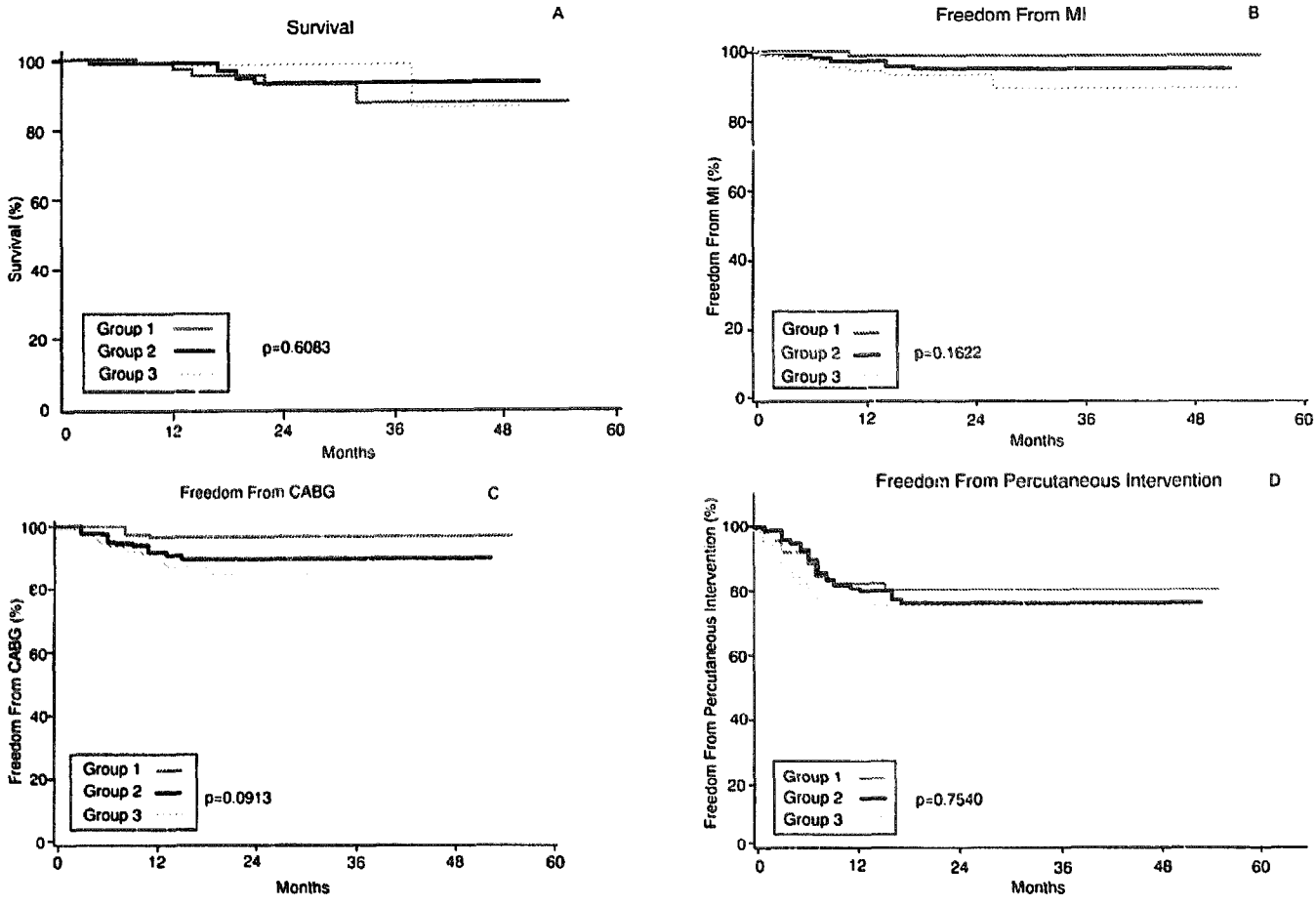
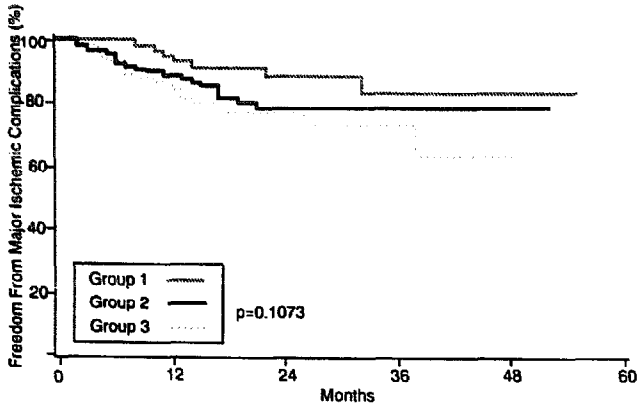



**Figure 1.** Plots showing freedom from death (A), myocardial infarction (MI) (B), coronary artery bypass surgery (CABG) (C) and repeat percutaneous coronary revascularization (D) according to angina status.

higher incidence of hospital admission for angina in the unstable angina groups (p = 0.048). Figure 2 shows the event-free survival curves for the three groups. At the end of the follow-up period, 83% of patients in the group with stable angina were free of major ischemic events (death, myocardial infarction, coronary bypass) compared with 78% in Group II and 62% in Group III (p = 0.10). The divergence of the survival curves occurred in the 1st 12 to 18 months, followed by a leveling off later in the follow-up period. At the end of the follow-up period, 68% of patients in the stable angina group were free of chest pain compared with 52% in Group II and 49% in Group III (p = NS).

**Figure 2.** Plot showing freedom from cumulative incidence of death, myocardial infarction and bypass surgery according to angina status.




## Discussion

Percutaneous transluminal coronary angioplasty was developed and initially indicated for the treatment of selected patients with stable angina (24). The procedure was subsequently introduced for the treatment of unstable angina by Meyer et al. (25) and Williams et al. (26) in 1980. In the first National Heart, Lung, and Blood Institute Registry (27), the syndrome of unstable angina accounted for ~40% of all coronary angioplasty attempted. However, only a few years later, unstable angina accounted for more than half of all angioplasties in many centers (22,28–32).

**Results of coronary angioplasty in unstable angina.** The primary angiographic success rate of coronary angioplasty in the setting of unstable angina is similar to that after coronary angioplasty in patients with stable angina, but the major

complication rates are higher, often because of the development of abrupt thrombotic occlusion (32–34). Recent studies have shown that, even with current technology, coronary angioplasty-related major complications occur in 3% to 12% of patients with unstable angina (3,4,32).

*Coronary angioplasty–unstable angina interaction.* The higher complication rate associated with coronary angioplasty in unstable angina probably reflects the unfavorable interaction of angioplasty with the intracoronary thrombus presumed to be present in unstable angina. Angiographic evidence of intracoronary thrombus in patients with unstable angina ranges from 1% to 53%, depending on the definition of thrombus and the time elapsed between the onset of symptoms and angiography (35–40). Moreover, recent angioscopic studies have shown the low sensitivity (as low as 14%) of angiography in detecting intracoronary thrombus (41,42). This is especially true with complex lesions and in acute ischemic syndromes (unstable angina, acute myocardial infarction, postinfarction angina) (43). Indeed, the frequency of thrombus occurrence with the syndrome approaches 100% when diagnostic techniques such as angioscopy are used (44). Moreover, coronary angioplasty per se might aggravate thrombus formation. Animal studies have shown that coronary angioplasty causes immediate platelet deposition, mural thrombosis and localized constriction at the site of arterial injury (45–48). In animal models, angioplasty has been shown to reduce the maximal hyperemic coronary blood flow (49), and in humans, coronary blood flow reserve is often depressed immediately after angioplasty (50).

In unstable coronary syndromes, by mechanically disrupting an existing fresh intraarterial thrombus, coronary angioplasty causes release of vasoactive substances, which results in downstream microvascular constriction (51,52). Coronary angioplasty can also increase the risk of embolizing atheromatous or thrombotic debris, especially after dilation after recent acute myocardial infarction (53,54). Several studies have shown that angioplasty of thrombus-containing lesions is associated with an increased risk of acute thrombotic coronary occlusion (up to 73% of patients in older series), with subsequent increased incidence of death, myocardial infarction and emergency surgical intervention (5–11). Even with routine antiplatelet therapy and heparinization in this setting, there is still a twofold increase in the risk of ischemic complications in this setting (6,7).

**Theoretical appeal of directional atherectomy.** Plaque excision by directional atherectomy could be beneficial in the setting of unstable angina by interrupting the vicious cycle whereby thrombus begets more thrombus. In fact, directional atherectomy has been used to treat abrupt occlusion by a debulking effect, that is, by excising an occluding intraluminal dissection or intraluminal defect superimposed on atheromatous tissue (55,56). Hinohara et al. (15) demonstrated that directional coronary atherectomy is an effective and safe treatment for angiographically complex lesions that are less appropriate for coronary angioplasty. Popma et al. (21) found that the procedural results of directional coronary atherectomy were not adversely affected by the presence of thrombus or lesion irregularity. Ellis et al. (17) also found that complex, thrombus-associated stenoses have a favorable outcome with directional coronary atherectomy. It was suggested that the relatively soft nature of the stenosis and the removal of the nidus for thrombus formation by atherectomy makes such lesions particularly well suited to treatment with directional atherectomy (17).

**Immediate results.** Previous studies have shown that directional coronary atherectomy can be performed successfully and safely despite its frequent use in lesions that are considered high risk for coronary angioplasty (14–17,21,57). Our procedural success rate is consistent with the results reported from other series. The incidence of major ischemic complications was higher in patients with rest or postinfarction angina, or both, compared with the other two groups, which were nearly identical. These results point to the fact that the definition of unstable angina is important in assessing the immediate outcome of directional coronary atherectomy because in the absence of rest or postinfarction angina, there was no difference in the immediate complication rate between stable and unstable angina. These results are similar to those previously reported for coronary angioplasty in unstable angina by de Feyter et al. (3), who also showed that the definition of unstable angina predicts the outcome of coronary angioplasty in this setting and that in the absence of rest or postmyocardial infarction angina, the results of angioplasty are not significantly different from those obtained in a population with stable angina. The results also demonstrate that the use of directional coronary atherectomy in this setting is not without complications and that the simplistic view that directional atherectomy removes the unstable plaque and therefore prevents the immediate complications associated with such plaque is in error. However, this study does not address whether directional coronary atherectomy is a better technique than angioplasty in dealing with unstable plaque in the setting of rest or postinfarction angina, or whether a period of stabilization with heparin before directional coronary atherectomy may improve the results. Our results are consistent with the hypothesis that the immediate outcome in unstable angina is not affected by the device used (in this case the Atherocath) but rather by the underlying pathophysiology and that directional atherectomy does not solve the problem of unstable plaque in patients with rest or postinfarction angina.

The only other report that specifically addressed the role of directional coronary atherectomy in unstable angina was that of the Sequoia group in abstract form (58). This report, which addressed only the immediate results of atherectomy in unstable angina, examined 452 patients, approximately half of whom presented with unstable angina. This group reported a success rate of 92%, emergency bypass in 5.6%, Q wave myocardial infarction in 2.4% and CK elevation in 19% for the unstable angina group compared to 96%, 2.5%, 0% and 12%, respectively, for the stable angina group.

JACC Vol. 24, No. 1
July 1994:46–54

Similar to our results, when the group with the severe forms of unstable angina were reviewed separately, these investigators found a higher incidence of immediate complications, especially after emergency bypass surgery (10%).

**Clinical follow-up.** Despite the equal distribution of left ventricular dysfunction and multivessel disease among the three groups, the event-free survival rate curves show an initial divergence in the 1st 12 months. This divergence probably reflects the natural progress of unstable angina and was followed by a flattening of the curves between 12 and 18 months, with a steady parallel decline in event-free survival rate. This steady and parallel decline probably represents the natural history of patients with complex coronary artery disease. After the first year, there was a death rate of 3% to 5%/year and a 10%/year incidence of major ischemic events (myocardial infarction, coronary bypass, angioplasty). Fishman et al. (14) observed a similar pattern and found that most of the adverse effects after directional atherectomy occur within the 1st 6 months (12% bypass, 17% angioplasty) (14). Beyond the first follow-up year, they also observed a 5%/year death rate and a 7%/year ongoing repeat event rate during the second and third year after directional atherectomy.

**Comparison with previous coronary angioplasty studies.** Although a direct comparison with previous coronary angioplasty series is difficult, the overall survival and event-free survival rates compare favorably with those reports in similar patients after conventional coronary angioplasty from the National Heart, Lung, and Blood Institute Registry (22). These investigators reported a cumulative freedom from major ischemic events (death, infarction, coronary bypass, repeat angioplasty) of 64% for patients with stable angina and 61% for patients with unstable angina at 2 years. Here, again, most of the adverse effects after angioplasty occurred within the 1st 12 months, with the survival rate curves leveling off between 12 and 18 months (angioplasty 18%, bypass 12% at 1 year).

**Study limitations.** This study was not a randomized treatment evaluation, and caution must therefore be urged in the comparison of these results with those achieved by other technologies. The patients in this study may differ significantly from typical patients undergoing angioplasty. Many patients with unfavorable lesion characteristics, such as tortuous vessels, severely angulated lesions, diffusely diseased vessels and heavily calcified lesions, were excluded because the use of directional coronary atherectomy was believed to be contraindicated in our center. Moreover, the duration of postatherectomy heparinization and the use of postatherectomy balloon dilation were not predefined and were left to the interventionalist's discretion. It is possible that a prolonged postatherectomy heparinization period or an aggressive postatherectomy balloon dilation may have improved the results, especially in Group III.

The study patients included only those who had a directional coronary atherectomy for a single de novo stenosis. Although limiting the study in such a way was necessary to avoid the effects of other confounding variables that can potentially influence the clinical outcome (such as restenotic lesions, use of more than one device in a single patient), it makes the generalization of the results of this study to all groups not necessarily valid. Indeed, the slightly higher immediate complication rates in our series could be related to the inclusion of only de novo lesions, which have been shown to have a higher incidence of abrupt closure (59). Moreover, because of the small number of vein grafts treated by atherectomy in this series, we did not analyze this subgroup separately. There was no death, Q myocardial infarction or emergency bypass surgery in this small subgroup. However, because of the small number, we cannot generalize our findings to the treatment of vein grafts by atherectomy.

The study also included early and late experience with the atherectomy device, encompassing a period when operators were gaining more experience and when there were slight modifications of the atherectomy device. These factors could have an impact on the immediate results of the device, but this is unlikely because the complication rates were equally distributed between the old and new experience, again strengthening the argument that it is the pathophysiology of unstable angina rather than the mechanical intervention per se that determines the immediate and short-term outcome.

**Conclusions.** This is the first report comparing the immediate and follow-up results of directional coronary atherectomy in patients with stable versus unstable angina. The results reported herein suggest that directional atherectomy may be applied in patients with unstable angina with acceptable success and complication rates. The procedural success and complication rates are similar for patients with stable versus those with unstable angina who do not present with rest or postinfarction angina. The immediate complication rates are higher in the subgroup of patients with unstable angina presenting with rest or postinfarction angina, or both. The lower event-free survival rate, especially in the first year after the procedure, most likely represents the underlying pathophysiology and natural history of unstable angina. Long-term events after the procedure seem to reflect the natural history of coronary artery disease. Major impact on the results of percutaneous interventions in patients with rest or postinfarction angina may only be achieved by improved pharmacologic adjuncts to inhibit platelet aggregation (60) or thrombus formation (61), or both.

We gratefully acknowledge Deborah Lynch, Gan Howell, Freddie Ford, J. Patrick Lang and all the staff of the Interventional Registry at The Cleveland Clinic Foundation for their effort in the collection of data and Kathryn Brock for editorial assistance.

## References

1. Ross AM. The role of coronary angioplasty in the management of unstable angina pectoris. Am J Cardiol 1991;68 Suppl:58C–60C.

2. Faxon DP. Percutaneous coronary angioplasty in stable and unstable angina. Cardiol Clin 1991;9:99–114.
3. de Feyter PJ, Serruys PW, van den Brand M, Hugenholtz PG. Percutaneous transluminal coronary angioplasty for unstable angina. Am J Cardiol 1991;68 Suppl:125B–35B.
4. Myler RK, Shaw RE, Stertzer SH, et al. Unstable angina and coronary angioplasty. Circulation 1990;82 Suppl II:II-88–95.
5. Mabin TA, Holmes DR, Smith HC. Intracoronary thrombus role in coronary occlusion complicating percutaneous transluminal coronary angioplasty. J Am Coll Cardiol 1985;5:198–202.
6. Sugrue DR, Holmes DR, Smith HC. Coronary artery thrombus as a risk factor for acute vessel occlusion during percutaneous transluminal coronary angioplasty: improved results. Br Heart J 1986;56:62–6.
7. Deligonul U, Gabliani GI, Caroles DG, et al. Percutaneous transluminal coronary angioplasty in patients with intracoronary thrombus. Am J Cardiol 1988;62:474–6.
8. Mooney MR, Fishman-Mooney J, Goldenberg I, et al. Percutaneous transluminal coronary angioplasty in the setting of large intracoronary thrombus. Am J Cardiol 1990;65:427–31.
9. Schieman G, Cohen BM, Kozina J, et al. Intracoronary urokinase for intracoronary thrombus accumulation complicating percutaneous transluminal coronary angioplasty in acute ischemic syndromes. Circulation 1990;82:2052–60.
10. Ellis SG, Roubin GS, King SB, et al. Angiographic and clinical predictors of acute closure after native vessel coronary angioplasty. Circulation 1988;77:372–9.
11. Detre KM, Holmes DR, Holubkov R, et al. Incidence and consequences of periprocedural occlusion. The 1985–1986 National Heart, Lung and Blood Institute PTCA registry. Circulation 1990;82:739–50.
12. Chesebro JH, Badimon L, Fuster V. Importance of antithrombin therapy during coronary angioplasty. J Am Coll Cardiol 1991;17 Suppl:96B–100B.
13. Zeiher AM, Schachinger V, Weitzel SH, et al. Intracoronary thrombus formation causes focal vasoconstriction of epicardial arteries in patients with coronary artery disease. Circulation 1991;83:1519–25.
14. Fishman RF, Kuntz RE, Carrozza JP, et al. Long-term results of directional coronary atherectomy: predictors of restenosis. J Am Coll Cardiol 1992;20:1101–10.
15. Hinohara T, Rowe MH, Robertson GC, et al. Effect of lesion characteristics on outcome of directional coronary atherectomy. J Am Coll Cardiol 1991;17:1112–20.
16. Hinohara T, Selmon MR, Robertson GC, et al. Directional atherectomy: new approaches for treatment of obstructive coronary and peripheral vascular disease. Circulation 1990;81 Suppl IV:IV-79–91.
17. Ellis SG, De Cesare NB, Pinkerton CA, et al. Relation of stenosis morphology and clinical presentation to the procedural results of directional coronary atherectomy. Circulation 1991;84:644–53.
18. Ellis SG, Vandormael MG, Cowley MJ, et al. Coronary morphologic and clinical determinants of procedural outcome with angioplasty for multivessel coronary disease: implications for patient selection. Circulation 1990;82:1193–202.
19. Bell MR, Bailey KR, Reeder GS, Lapeyre AC III, Holmes DR Jr. Percutaneous transluminal coronary angioplasty in patients with multivessel disease: how important is complete revascularization for cardiac event-free survival? J Am Coll Cardiol 1990;16:553–62.
20. Dorros G, Cowley MJ, Simpson J, et al. Percutaneous coronary angioplasty: report of complications from the National Heart, Lung, and Blood Institute PTCA Registry. Circulation 1983;67:723–30.
21. Popma JJ, DeCesare NB, Ellis SG, et al. Clinical, angiographic and procedural correlates of quantitative coronary dimensions after directional coronary atherectomy. J Am Coll Cardiol 1991;18:1183–9.
22. Bentivoglio LG, Holubkov R, Kelsey SF, et al. Short and long term outcome of PTCA in unstable angina pectoris: a report of the 1985–1986 NHLBI PTCA Registry. Cathet Cardiovasc Diagn 1991;23:227–38.
23. Topol EJ, Leya F, Pinkerton CA, et al. A comparison of directional atherectomy with coronary angioplasty in patients with coronary disease. N Engl J Med 1993;329:221–7.
24. Gruentzig AR, Senning A, Siegenthaler WE. Non operative dilatation of coronary artery stenosis: percutaneous transluminal coronary angioplasty. N Engl J Med 1979;301:61–8.
25. Meyer J, Schmitz HJ, Kiesslich T, et al. Percutaneous transluminal coronary angioplasty in patients with stable and unstable angina pectoris: analysis of early and late results. Am Heart J 1983;106:973–80.
26. Williams DO, Riley RS, Singh AK, Gewirtz H, Most AS. Evaluation of the role of coronary angioplasty in patients with unstable angina pectoris. Am Heart J 1981;102:1–9.
27. Detre KM, Myler RK, Kelsey SF, Van Raden M. Baseline characteristics of patients in the National Heart, Lung and Blood Institute percutaneous transluminal coronary angioplasty registry. Am J Cardiol 1984;53 Suppl: 7C–11C.
28. Faxon DP, Detre KM, McCabe CH, et al. Role of percutaneous transluminal coronary angioplasty in the treatment of unstable angina. Am J Cardiol 1983;53 Suppl:131C–5C.
29. Steffenino G, Meier B, Finci L, Rutishauser W. Follow up results of treatment of unstable angina by coronary angioplasty. Br Heart J 1987; 57:416–9.
30. Sen S, Ozbek C, Berg G, Bach R, Dyckmans J, Schieffer H. Treatment of unstable angina pectoris (European experience). Am J Cardiol 1991;68 Suppl:47C–51C.
31. Rupprecht HJ, Brennecke R, Kottmeyer M, Bernhard G, Erbel R, Pop T, Meyer J. Short- and long-term outcome after PTCA in patients with stable and unstable angina. Eur Heart J 1990;11:964–75.
32. de Feyter PJ, Suryapranata H, Serruys PW, et al. Coronary angioplasty for unstable angina: immediate and late results in 200 consecutive patients with identification of risk factors for unfavorable early and late outcome. J Am Coll Cardiol 1988;12:324–33.
33. Faxon DP, Kelsey SF, Ryan TJ, et al. Determinants of percutaneous transluminal angioplasty: report from the National Heart, Lung, and Blood Institute registry. Am Heart J 1984;108:1019–23.
34. Quigley PJ, Erwin J, Maurer BJ, Walsh MJ, Gearty GF. Percutaneous transluminal coronary angioplasty in unstable angina: comparison with stable angina. Br Heart J 1986;55:227–30.
35. Capone G, Wolf NM, Meyer B, Meister SG. Frequency of intracoronary filling defects by angiography in angina pectoris at rest. Am J Cardiol 1985;56:403–6.
36. Vetrovec GW, Cowley MJ, Overton H, Richardson DW. Intracoronary thrombus in syndromes of unstable myocardial ischemia. Am Heart J 1981;102:1202–8.
37. Zack PM, Ischinger T, Aker VT, Dineer B, Kennedy HL. The occurrence of angiographically detected intracoronary thrombus in patients with unstable angina pectoris. Am Heart J 1984;108:1408–12.
38. Holmes DR, Hartzler GO, Smith HC, Fuster V. Coronary artery thrombus in patients with unstable angina. Br Heart J 1981;45:411–6.
39. Freeman MR, Williams AE, Chisholm RJ, Armstrong RW. Intracoronary thrombus and complex morphology in unstable angina: relation to timing of angiography and in-hospital cardiac events. Circulation 1989;80:17–23.
40. Rehr R, DiSciascio G, Vetrovec G, et al. Angiographic morphology of coronary artery stenoses in prolonged rest angina: evidence of intracoronary thrombus. J Am Coll Cardiol 1989;14:1429–37.
41. Ramee SR, White CJ, Collins TJ, et al. Percutaneous angioscopy during coronary angioplasty using a steerable microangioscope. J Am Coll Cardiol 1991;17:100–5.
42. Mizuno K, Satumora K, Miyamoto A, et al. Angioscopic evaluation of coronary artery thrombi in acute coronary syndromes. N Engl J Med 1992;326:287–91.
43. Christou CP, Hatt JI, Goldstein JE, et al. Correlation of ischemic coronary syndromes with angiographic morphology and lesion histology [abstract]. Circulation 1992;86 Suppl I:I-731.
44. Sherman CT, Litvak F, Grundfelt W, et al. Coronary angioscopy in patients with unstable angina pectoris. N Engl J Med 1986;315:913–9.
45. Wilentz JR, Sanborn TA, Haudenschild CC, Valeri CR, Ryan T, Faxon DP. Platelet accumulation in experimental angioplasty: time course and relation to vascular injury. Circulation 1987;73:636–42.
46. Pasternak RC, Baughman KL, Fallon JT, Block PC. Scanning electron microscopy after coronary transluminal angioplasty of normal coronary arteries. Am J Cardiol 1980;45:591–8.
47. Lam JYT, Chesebro JH, Steele PM, Badimon L, Fuster V. Is vasospasm related to platelet deposition? Relationship in a porcine preparation of arterial injury in vivo. Circulation 1987;73:243–8.
48. Brezinski DA, Nesto RW, Serhan CN. Angioplasty triggers intracoronary leukotrienes and lipoxin A4. Impact of aspirin therapy. Circulation 1992;86:56–63.
49. Bates ER, McGillem MJ, Beals TF, et al. Effect of angioplasty-induced endothelial denudation compared with medial injury on regional coronary blood flow. Circulation 1987;76:710–6.

50. Wilson RF, Johnson MR, Marcus ML, et al. The effect of coronary angioplasty on coronary flow reserve. Circulation 1988;77:873–85.
51. Wilson RF, Lesser JR, Laxson DD, et al. Intense microvascular constriction after angioplasty of acute thrombotic coronary arterial lesions. Lancet 1989;1:807–11.
52. Babbitt DG, Perry JM, Forman MB. Intracoronary verapamil for reversal of refractory coronary vasospasm during percutaneous transluminal coronary angioplasty. J Am Coll Cardiol 1988;12:1377–81.
53. Bredlau CE, Roubin GS, Leimgruber PP, et al. In-hospital morbidity and mortality in patients undergoing elective coronary angioplasty. Circulation 1985;72:1044–52.
54. Colavita PG, Ideker RE, Reimer HA, et al. The spectrum of pathology associated with percutaneous transluminal angioplasty during acute myocardial infarction. J Am Coll Cardiol 1986;8:855–60.
55. McCluskey E, Cowley MJ, Whitlow PL. Directional coronary atherectomy for failed percutaneous transluminal coronary angioplasty. Am J Cardiol 1993;72 Suppl:42E–46E.
56. Hofling B, Gonschior P, Simpson L, et al. Efficacy of directional coronary atherectomy in cases unsuitable for percutaneous transluminal coronary angioplasty (PTCA) and after unsuccessful percutaneous transluminal coronary angioplasty. Am Heart J 1992;124:341–8.
57. Safian RD, Gelbfish JS, Erny RE, Senitt SJ, Schmidt DA, Baim DS. Coronary atherectomy. Clinical, angiographic, and histological findings and observations regarding potential mechanisms. Circulation 1990;82: 69–79.
58. Robertson GC, Hinohara T, Vetter JW, et al. Directional coronary atherectomy for the treatment of unstable angina [abstract]. J Am Coll Cardiol 1993;21:33A.
59. Popma JJ, Topol EJ, Hinohara T, et al. Abrupt vessel closure after directional coronary atherectomy. J Am Coll Cardiol 1992;19:1372–9.
60. Simoons ML, van den Brand M, Hoorntje JCA, et al. Chimeric 7E3 antiplatelet antibody Fab for treatment of refractory unstable angina: a placebo-controlled pilot study [abstract]. J Am Coll Cardiol 1993;21: 269A.
61. Lidon RM, Adelman B, Maraganon J, Theroux P. Hirulog, a direct thrombin inhibitor for the management of unstable angina [abstract]. Circulation 1992;86 Suppl I:I-386.